Pathogenesis of viruses pdf

Go to the main content See also: Introduction to viruses Viral pathogenesis is the study of the process and mechanisms by which viruses cause diseases in their target hosts, often at the cellular or molecular level. This is a specialized field of research in virology. Pathogenesis is a qualitative description of the process by which the initial infection causes the disease. Viral disease is the sum of the effect of viral replication on the host and the subsequent immune response of the host against the virus. Viruses are able to initiate infection, dissipate throughout the body and multiply due to specific virulence factors. There are several factors that affect pathogenesis. Some of these factors include the virulence of the characteristic virus that infects. In order to cause the disease, the virus must also overcome several inhibitory effects present in the host. Some of the inhibitory effects include distance, physical barriers and host protection. These inhibitory effects may differ among humans due to inhibitory effects genetically controlled. Viral pathogenesis depends on various factors: (1) transmission, input and spread within the host, (2) tropism, (3) virality virus and disease mechanisms, (4) host factors and host defense. The mechanisms of infectious viruses need to establish infections in host cells in order to multiply. For infections to occur, the virus must capture the host's host's host's host's immune response for effective replication. Viral replication often requires complex interaction between the virus and the host factors, which can lead to detrimental effects on the host, which gives the virus its pathogenicity. Important steps in the virus's life cycle that form the pathogenesis See also: Viral life cycle Typical places of virus in entry into the body: The first steps of a viral infection are determined by the place at which the virus implants into the body. This will subsequently dictate the mechanisms of viral pathogenesis. Transmission from host with infection to the second host Virus entry into the body Local replication in susceptible cells Spread and spread to secondary tissues and target organs Secondary replication in the susceptible cells spilling the virus into the environment Forward transmission to the third host survey of the main manifestations of viral infection6 Primary transmission 3 requirements must be satisfied to ensure successful host infection. First, you need to have enough virus to trigger the infection. Cells at the site of infection should be available, in that their cell membranes display host-coded receptors that the virus can use to enter the cell, and the receiving antiviral protection systems must be ineffective or absent. [3] to accommodate viruses that cause disease in humans often passes through the mouth, nose, sexual pathways, or damaged areas of the skin, so the cells of the respiratory, gastrointestinal, skin and genital tissues are often the main place of infection. Some viruses are capable of transmitting mammalian fetuses through infected germ cells during fertilization, later during pregnancy through the placenta, and by infection at birth. Localized replication and spread After initially entering the host, the virus captures the host cell's equipment to undergo viral gain. Here, the virus must modulate the host's innate immune response to prevent the body from eliminating it, making it easier to replicate. The replicated virus from the originally infected cell is then dissipated to infect neighboring susceptible cells, possibly with spread to different cell types such as white blood cells. This leads to a localized infection in which the virus mainly spreads and infects neighboring cells to the point of entry. Otherwise, the virus may be released into extracellular fluids. Examples of localized infections include colds (rhinovirus), influenza (parainfluenza), gastrointestinal infections (rotavirus) or skin infections (papillomavirus). Spread and re-replication In other cases, the virus can cause systemic disease through a common infection spread throughout the body. The predominant way the virus spreads occurs through the blood or lymphatic system, some of which include viruses responsible for chickenpox (chickenpox virus), smallpox (variol), HIV (human immunodeficiency virus). A minority of viruses can spread through the nervous system. It is noteworthy that poliovirus can be transmitted along the fecal-oral route, where it initially reproduces at the entrance point, the small intestine and spreads to regional lymph nodes. The virus then spreads through the bloodstream to various organs of the body (e.g. liver, spleen), followed by a secondary round of replication and spread in the central nervous system to damage motor neurons. Shed and secondary transmission Finally, viruses spread to sites where shedding in the environment can occur. Respiratory, alimony and urogenital tracts and blood are the most frequent places of shedding in the form of bodily fluids, aerosols, skin, excrement. The virus will then be transmitted to another person and re-establish the cycle of infection. Factors influencing pathogenesis There are several major comprehensive factors influencing viral disease: Virus tropism virus factors Host factors the Molecular Basis of Viral Tropism Virus Tropism refers to the virus's preferential replication site in discrete cell types in the organ. In most cases, tropism is determined by the ability of viral surface proteins to merge or bind to surface specific target cells to establish infection. Thus, linking the specifics of the viral surface of the surface dictates tropism, as well as the destruction of certain cell populations, and is therefore the main determinant of the pathogenesis of the virus. However, co-receptors are sometimes required in addition to binding cell receptors on host cells to viral proteins in order to establish infection. For example, HIV-1 requires target cells to express co-receptors CCR5 or CXCR4, on top of the CD4 receptor for productive viral attachment. Interestingly, HIV-1 can pass the tropism switch where the glycoprotein virus gp120 initially uses CCR5 (mainly on macrophagues) as the main joint receptor for entry into the host cell. Subsequently, HIV-1 switches to bind to CXCR4 (mostly on T cells) as the infection progresses, while transferring viral pathogens to another stage. In addition to cellular receptors, viral tropism can also be regulated by other intracellular factors, such as specific transcription factors. An example would be the JC poliovirus, in which its trocy is limited to glial cells, as its amplifier is only active in glial cells, and the expression of the JC viral gene requires host transcription factors expressed exclusively in glial cells. The accessibility of tissues and host organs to the virus also regulates tropism. Accessibility depends on physical barriers such as enteroviruses, which multiply in the gut, as they are able to withstand bile, digestive enzymes and acidic environments. Viral factors of viral genetics encoding viral factors will determine the degree of viral pathogenesis. This can be measured as virulence, which can be used to compare the quantitative degree of pathology between related viruses. In other words, different strains of viruses with different viral factors can lead to varying degrees of virulence, which in turn can be used to study differences in pathogenesis of viral variants with varying virulence. Viral factors are greatly influenced by viral genetics, which is a determining factor in the virulence of structural or non-structural proteins and non-coding sequences. In order for the virus to successfully infect and cause disease in the host, it must encode specific viral factors in its genome to overcome the preventive effects of physical barriers, and modulate the inhibition of host virus replication. In the case of poliovirus, all strains of vaccine found in oral polio vaccines contain mutations in 5' untranslated region (5' UTR). Conversely, the virulent strain responsible for causing polio does not contain these 5' UTR mutations and thus exhibits a large viral pathogenicity in the hosts. Viral factors encoded in the genome often control entry, shedding and transmission routes. It is believed that in polioviruses, mutations cause replication and translation defect to reduce the decrease in the ability of the virus to cross-referencing host cells and replication in the nervous system. Viruses have also developed various immunomodulation mechanisms to undermine the host's immune response. It is usually have virus-coded bait receptors that target cytokines and chemokines produced as part of the host's immune response, or homologies of the host's cytokines. Thus, viruses that can manipulate the reaction of host cells to infection as a strategy to evade immunity exhibit greater pathogenicity. The host factors of viral pathogenesis also depend heavily on the taking factors. Several viral infections have shown different effects, ranging from imptomatic to symptomatic or even critical infection, solely based on various host factors alone. In particular, genetic factors, age and immunocompetence play an important role in dictating whether a viral infection can be modulated by the host. Mice with functional Mx genes encode the Mx1 protein, which can selectively inhibit influenza replication. Thus, mice carrying a non-functional Mx allele are unable to synthesize the Mx protein and are more susceptible to influenza infection. Alternatively, individuals with weakened immunity due to existing diseases may have a defective immune system, making them more vulnerable to the virus's damage. In addition, a number of viruses display variable pathogenicity depending on the age of the host. Pig, polio and the Epstein-Barr virus cause more severe diseases in adults, while others, such as rotavirus, cause more severe infection in infants. Therefore, it is assumed that the host's immune system and defense mechanisms may differ with age. Disease Mechanisms: How Do Viral Infections Cause Diseases? Viral infection does not always cause disease. A viral infection simply involves viral replication in the host, but the disease damages the viral multiplication. A person who has a viral infection but shows no symptoms of the disease is known as a carrier. The mechanisms by which viruses cause harm and disease to host cells damage the virus Caught inside the host cells, viruses can destroy cells through various mechanisms. Viruses often cause direct cytopal effects to disrupt cellular function. This can be by releasing enzymes to degrade the host's metabolic precursors, or to release proteins that inhibit the synthesis of important host factors, proteins, DNA and/or RNA. Specifically, the viral proteins of the herpes simplex virus can degrade the host's DNA and inhibit the DNA of the host cell and the transcription of mRNA. The poliovirus can inactivate the proteins involved in the transfer of host mRNA without affecting the translation of poliovirus mRNA. In the the expression of viral fusion proteins on the surface of host cells can lead to the synthesis of host cells that form multi-celled cells. Notable examples include measles measles HIV, respiratory syncytial virus. Virus lifestyle strategies in host cells. Acute infections usually occur within a relatively short period of time, while persistent infections when the virus is not completely cleared of the body. In hidden infections, reactivation of the disease can occur for a long time after the initial infection. It is important to note that viral infections may differ from lifestyle strategies. Persistent infections occur when cells continue to survive despite a viral infection and can be further classified into hidden (only the viral genome is present, no replication occurs) and chronic (basal levels of viral replication without stimulating the immune response). In acute infections, lytic viruses shed on high for a rapid infection of secondary tissue/host, while persistent viruses undergo shedding on lower over a longer transmission period (from months to several years). Lithiuc viruses are capable of destroying host cells by taking on and/or interfering with the specialized functions of host cells. An example would be the launch of necrosis in host cells infected with the virus. Otherwise, signature viral infections, such as binding HIV to co-receptors CCR5 or CXCR4, can also cause cell death through apoptosis through the receiving signaling cascades of immune cells. However, many viruses encode proteins that can modulate apoptosis depending on whether the infection is acute or permanent. Induction of apoptosis, for example, through interaction with caspase, will promote viral spilling of lytic viruses to facilitate transmission, while viral inhibition of apoptosis can prolong the production of the virus in cells, or allow the virus to remain hidden from the immune system in chronic, persistent infections. However, induction of apoptosis in major immune cells or antigen-representing cells may also act as a mechanism for immunosuppression in persistent infections such as HIV. The main cause of immunosuppression in HIV-infected patients is the depletion of CD4'T-assistant cells. Interestingly, adenovirus has the protein E1A to cause apoptosis, initiating a cell cycle, and the protein E1B to block the apoptotic pathway through inhibition of caspas interaction. Persistent viruses can sometimes convert host cells into cancer cells. Viruses such as human papillomavirus (HPV), human T-lymphotropic virus (HTLV), etc., can stimulate the growth of tumors in infected hosts, either by disrupting the expression of the tumor suppressor gene (HPV), or by re-phoning proto-oncogenic expression (HTLV). Damage caused by the host's immune system Sometimes, instead of cell death or cell dysfunction, virus, the host's immune response can be a mediator in disease and excessive inflammation. Stimulation of the congenital and adaptive immune system in response to viral infection destroys cells, which can lead to severe pathological consequences for the host. This damage to the immune system is known as immunopathology caused by the virus. In particular, immunopathology is caused by the excessive release of antibodies, interferons and pro-inflammatory cytokines, the activation of the supplement system or hyperactivity of cytotoxic T cells. The secretion of interferons and other cytokines can cause cell damage, fever and flu-like symptoms. In severe cases of some viral infections, as in avian influenza H5N1 in 2005, abnormal induction of the host's immune response can cause the burning of the release of cytokines, known as cytokine storm. In some cases, a viral infection can trigger an autoimmune response that arises through various proposed mechanisms: molecular mime and observer mechanism. Molecular mimimus refers to overlapping the structural similarity between the viral antigen and the antigen itself. The observer mechanism involves initiating a non-specific and excessive antiviral response that solves the problems of self-criticism in the process. Damage caused to the owner due to autoimmune diseases was observed in the West Nile virus. Incubation viruses display variable incubation periods when the virus enters the host. The incubation period refers to the time it takes to start a disease after the first contact with the virus. The incubation period varies from the distance traveled by the virus to the target organ; but in most viruses, the duration of incubation depends on many factors. Surprisingly, generalized togavirus infections have a short incubation period due to the direct penetration of the virus into target cells through insect bites. There are several other factors that affect the incubation period. Mechanisms behind long incubation, months or years, for example, are not yet fully understood. Evolution of Virulence Some relatively ayirulent viruses in their natural host show increased virulence when moving to a new host species. When the emerging virus first invades a new host species, the hosts have little or no immunity to the virus and often suffer from high mortality. Over time, there is sometimes a decrease in virulence in the prevailing deformation. A successful pathogen should spread to at least one other host, and lower virulence may lead to higher rates of transmission in certain circumstances. Similarly, genetic resistance to the virus can develop in host populations over time. An example of the evolution of virulence in the nascent virus is the case of myxomatose in rabbits. The release of wild European rabbits in 1859 in Victoria, Australia for sport to the rabbit's plague. In order to curb rabbit overcrowding, the virus mixoma, a deadly species-specific smallpox virus responsible for myxomatosis in rabbits, rabbits, intentionally released in south Australia in 1950. This reduced rabbit populations by 90%, and the disease became endemic within five years. It is noteworthy that severely weakened strains of the mystoma virus were detected in just 2 years of its release, and the genetic resistance in rabbits appeared within seven years. 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